In the United States Court of Federal Claims

OFFICE OF SPECIAL MASTERS

Filed: March 20, 2025

ALEXZANDER ROSE,	*	No. 17-1770V
Petitioner,	*	Special Master Young
v.	*	
SECRETARY OF HEALTH AND HUMAN SERVICES,	*	
Respondent.	*	

Mark Theodore Sadaka, Law Offices of Sadaka Associates, LLC, Englewood, NJ, for Petitioner.
Meghan Murphy, U.S. Department of Justice, Washington, DC, for Respondent.

ENTITLEMENT DECISION[1]

On November 13, 2017, Denise Sheffield-Rose and Mark Rose filed a petition for compensation in the National Vaccine Injury Compensation Program ("the Program")[2] on behalf of their then-minor son Alexzander Rose ("Petitioner").[3] Pet., ECF No. 1. Petitioner's parents alleged the human papillomavirus ("HPV") vaccine Petitioner received on March 30, 2015, caused him to suffer from alopecia areata ("AA"). *Id.* After carefully analyzing and weighing all the evidence and testimony presented in this case in accordance with the applicable legal standards,[4] I find that Petitioner has failed to provide preponderant evidence that the HPV vaccine he received

[1] Because this Decision contains a reasoned explanation for the action taken in this case, it must be made publicly accessible and will be posted on the United States Court of Federal Claims' website, and/or at https://www.govinfo.gov/app/collection/uscourts/national/cofc, in accordance with the E-Government Act of 2002. 44 U.S.C. § 3501 note (2018) (Federal Management and Promotion of Electronic Government Services). This means the Decision will be available to anyone with access to the internet. In accordance with Vaccine Rule 18(b), Petitioner has 14 days to identify and move to redact medical or other information, the disclosure of which would constitute an unwarranted invasion of privacy. If, upon review, I agree that the identified material fits within this definition, I will redact such material from public access.

[2] National Childhood Vaccine Injury Act of 1986, Pub L. No. 99-660, 100 Stat. 3755 ("the Vaccine Act" or "Act"). Hereinafter, for ease of citation, all "§" references to the Vaccine Act will be to the pertinent subparagraph of 42 U.S.C. § 300aa (2018).

[3] On January 24, 2025, the case caption was amended to reflect Petitioner's adult status. ECF No. 95.

[4] While I have reviewed all of the information filed in this case, only those filings and records that are most relevant to the decision will be discussed. *Moriarty v. Sec'y of Health & Hum. Servs.*, 844 F.3d 1322, 1328 (Fed. Cir. 2016) ("We generally presume that a special master considered the relevant record evidence even though he does not explicitly reference such evidence in his decision.") (citation omitted); *see also Paterek v. Sec'y of Health & Hum. Servs.*, 527 F. App'x 875, 884 (Fed. Cir. 2013) ("Finding certain information not relevant does not lead to—and likely undermines—the conclusion that it was not considered.").

on March 30, 2015, caused him to develop alopecia. Accordingly, Petitioner is not entitled to compensation.

I. Procedural History[5]

On November 13, 2017, Mark Rose and Denise Sheffield-Rose filed the petition. Pet. Petitioners filed medical records on November 14, 2017. Pet'r's Exs. 1–11, ECF Nos. 5–6. Petitioners filed two affidavits and a statement of completion on January 11, 2018. Pet'r's Exs. 12–13, ECF Nos. 14–15.

On April 19, 2018, Respondent filed his Rule 4(c) Report, recommending that compensation be denied. Resp't's Report, ECF No. 19. Petitioners submitted additional medical records on May 24, 2018, and June 11, 2018. Pet'r's Ex. 14, ECF No. 21; Pet'r's Ex. 15, ECF No. 22. On August 14, 2018, Petitioners filed an expert report from M. Eric Gershwin, M.D., M.A.C.P., M.A.C.R. as well as his curriculum vitae ("CV") and accompanying medical literature. Pet'r's Ex. 16, ECF No. 29; Pet'r's Exs. 27–97, ECF Nos. 31–37; Pet'r's Ex. 98, ECF No. 38. Petitioners filed additional medical literature on August 15, 2018. Pet'r's Exs. 17–26, ECF No. 41.

Respondent filed expert reports and CVs from Arnold I. Levinson, M.D., and Maryanne Makredes Senna, M.D., on May 8, 2019. Resp't's Exs. A–D, ECF No. 47. On March 9, 2020, Respondent filed a supplemental expert report from Dr. Levinson and the accompanying medical literature. Resp't's Ex. E, ECF No. 63; Resp't's Ex. C, Tabs 1–61, ECF Nos. 57–62. Petitioners filed a responsive expert report from Dr. Gershwin on March 27, 2020. Pet'r's Ex. 99, ECF No. 64. Petitioners submitted additional medical literature on March 30, 2020. Pet'r's Exs. 100–105, ECF No. 65.

On April 30, 2020, Respondent filed a second supplemental expert report from Dr. Levinson. Resp't's Ex. F, ECF No. 66. Petitioners filed a responsive supplemental expert report from Dr. Gershwin, along with accompanying medical literature, on September 14, 2020. Pet'r's Ex. 106, ECF No. 68; Pet'r's Exs. 107–115, ECF No. 69. Respondent filed additional medical literature on October 20, 2020. Resp't's Ex. A, Tabs 1–14, ECF Nos. 70–71. Respondent filed a third responsive supplemental expert report from Dr. Levinson on December 11, 2020. Resp't's Ex. G, ECF No. 75. Petitioners filed a final supplemental expert report from Dr. Gershwin and medical literature on June 25, 2021. Pet'r's Ex. 116, ECF No. 77; Pet'r's Exs. 117–123, ECF No. 78. Respondent filed an expert report from Emanual Maverakis, M.D., on March 27, 2023. Resp't's Ex. H, ECF No. 86. On February 5, 2024, Petitioners filed a motion for ruling on the record. Pet'r's Mot., ECF No. 92. Respondent filed his response on March 4, 2024; Petitioners replied on March 18, 2024. Resp't's Response, ECF No. 93; Pet'r's Reply, ECF No. 94. Petitioner reached the age of majority on November 15, 2019, and the caption was amended on January 24, 2025. ECF No. 95. This matter is ripe for consideration.

II. Factual Background

[5] Although Alexzander Rose is the captioned Petitioner as of January 24, 2025, prior to that date, his parents served as Petitioners on his behalf and were responsible for all filings. Therefore, throughout the procedural history section of this decision, Mark Rose and Denise Sheffield-Rose will be referred to as Petitioners to indicate that they completed the record while the captioned petitioner, Alexzander Rose, was still a minor. Alexzander Rose is the designated Petitioner in all other sections of this decision.

A. Medical Records

Petitioner was born on November 15, 2001. Pet'r's Ex. 3 at 1; ECF No. 5–3. On August 28, 2014, Petitioner was seen at Pediatric Health Care Alliance ("PHCA") for his annual health maintenance examination. *Id.* 3 at 7–9. He received a Tdap booster and his first HPV (Gardasil) and meningococcal vaccinations during this visit. *Id*. at 5. Petitioner returned to PHCA on November 3, 2014, and March 30, 2015, to receive his second and third HPV vaccinations. *Id*. at 38, 1.

Almost three months later, on June 12, 2015, Petitioner presented to Florida Acupuncture Solutions with a chief complaint of hair loss and allergies. Pet'r's Ex. 1 at 20, ECF No. 5. According to the visit notes, Ms. Sheffield-Rose noticed several bald spots on Petitioner's scalp after a haircut two weeks prior, but she had not noticed any hair loss after previous haircuts. *Id*. There was no history of stress or physical or emotional trauma, and the cause of Petitioner's hair loss was unclear. *Id*. Assessment notes indicated alopecia and a plan to administer a B12 injection. *Id*. Lab tests performed on June 16, 2015, for thyroid stimulating hormone ("TSH") levels, thyroglobulin antibodies, and thyroid peroxidase antibodies were unremarkable. Pet'r's Ex. 4 at 10, ECF No. 4.[6] Petitioner returned to Florida Acupuncture Solutions on June 29, 2015, July 3, 2015, July 14, and July 20, 2015, complaining of hair loss and allergies. Pet'r's Ex. 1 at 16–19, 22. Each visit noted alopecia under the assessment section. *Id.*

On July 15, 2015, Petitioner saw dermatologist Philip Barton, M.D., at Ocala Dermatology for AA on the scalp. Pet'r's Ex. 5 at 3, ECF No. 5. The history of present illness section noted that Petitioner "complained of hair loss that [was] multifactorial and moderate in severity." *Id*. Additional section notes indicated that Petitioner was using Clobetasol and Rogaine and had been given a Kenalog[7] injection four weeks prior. *Id.* Petitioner reported some hair regrowth to Dr. Barton. *Id*. On examination, Petitioner had "discrete, round, smooth to slightly shiny, alopecic patches" on the scalp. *Id*. Dr. Barton injected one lesion with Kenalog. *Id*. Petitioner was advised to contact Dr. Barton's office if his alopecia did not improve or if it worsened despite treatment. *Id*. On August 12, 2015, Petitioner visited with Ocala Dermatology to follow up on his AA. *Id.* at 1. Dr. Barton administered another Kenalog injection. *Id.*

On September 10, 2015, Petitioner presented to All Pediatrics complaining of AA that started in May 2015. Pet'r's Ex. 4 at 5–6. Visit notes showed that Petitioner reported receiving scalp steroid shots at New York City Dermatology and was given steroid lotions.[8] *Id.* at 6.

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[6] On June 22, 2015, Petitioner presented to Murray Hill Dermatology. Pet'r's Ex. 6 at 1, ECF No. 5–6. Visit notes were illegible; however, Respondent's brief stated that Petitioner "was assessed with alopecia areata ("AA") with hair loss on the scalp, but good hair in the eyebrows, eyelid, axilla, and pubic area." Resp't's Response at 4 (citing Pet'r's Ex. 6 at 3).

[7] Kenalog is a "trademark for preparations of triamcinolone acetonide." *Dorland's Illustrated Medical Dictionary* (33rd ed. 2020) [hereinafter "*Dorland's*"]. Triamcinolone acetonide "is applied topically to the skin or oral mucosa as an antiinflammatory and administered by inhalation for the chronic treatment of asthma, intranasally in the treatment of allergic rhinitis and other inflammatory nasal conditions, and by intra-articular, intradermal, intralesional, intramuscular, intrabursal, or tendon sheath injection as an antiinflammatory and immunosuppressant in a wide variety of disorders." *Id.*

[8] Petitioner did not submit records from the New York City dermatology visit.

Assessment notes listed the diagnosis as "alopecia totalis."[9] *Id.* Petitioner was encouraged to follow up with dermatology for alopecia treatments. *Id.* Additional notes also showed that Petitioner had sought a referral to "USF" for further evaluation and treatment.[10] *Id*.

Office notes from a February 8, 2016, visit to Pediatric Health Care Alliance referenced Petitioner's mother's statement that he received treatment at "NYU"[11] for alopecia, and that all testing was normal. Pet'r's Ex. 3 at 36. He was offered a referral for further treatment of his alopecia. *Id*.

B. Affidavits

In her affidavit filed on January 11, 2018, Ms. Sheffield-Rose stated that prior to developing alopecia, Petitioner "did not suffer from any chronic medical condition." Pet'r's Ex. 13 at ¶ 4, ECF No. 14-2. Ms. Sheffield-Rose recounted Petitioner's March 30, 2015 vaccination and recalled that she began noticing an unusual amount of hair in Petitioner's bathroom sink in mid to late April 2015. *Id.* at ¶¶ 5, 7. She noted that her sister visited on May 3, and June 9, 2015. *Id.* at ¶ 10. During the June visit, her sister commented that when she came in May, she noticed "the initial small bald spot, but did not mention anything to us." *Id.* Ms. Sheffield-Rose stated that she also "noticed a small bald spot on the top of [Petitioner's] head on or about May 4, 2015." *Id.* at ¶ 7. On May 18, 2015, Ms. Sheffield-Rose recounted that she "noticed another dime-sized bald spot on the top of [Petitioner's] head towards the back." *Id.*

Ms. Sheffield-Rose took Petitioner to Great Clips on May 31, 2015, where she noticed three quarter-sized bald spots toward the back of his head after his haircut. *Id.* at ¶ 9. She asked the hairdresser what happened, and the hairdresser "indicated that she did not know." *Id.* At that time, Ms. Sheffield-Rose believed that the hairdresser somehow made a mistake with a razor and "figured the hair would grow back soon." *Id.*

According to the affidavit, on June 29, 2015, Petitioner "began taking vitamins, airborne, emergen-C packs, and began applying Fluocinonide in his hair at night. He also used Clobetasol Propionate and Rogaine, recommended by Dr. Barton and the specialist at NYU." *Id.* at ¶ 13.

Ms. Sheffield-Rose described how Petitioner began to lose his body hair, and noted that by November 2015, he did not have hair on any part of his body. *Id.* at ¶ 17. Ms. Sheffield-Rose also noticed Petitioner's declining academic performance and behavioral changes. *Id.* at ¶ 19. Ms. Sheffield-Rose explained that Petitioner became "frustrated, non-social, and kept to himself." *Id.* She stated that Petitioner ultimately changed schools in 2016. *Id.* at ¶ 20.

III. Experts

A. Expert Qualifications

[9] Total alopecia, or alopecia totalis, is "complete loss of hair from the entire scalp, resulting from progression of alopecia areata." *Dorland's.*at 53.

[10] Neither Petitioner nor the medical records further clarified "USF," and no medical records from "USF" were filed. It is unclear if Petitioner was seen at USF or by any other dermatologist.

[11] Neither Petitioner nor the medical records further clarified "NYU," and no medical records from "NYU" were filed.

1. Petitioner's Expert, M. Eric Gershwin, M.D.

Dr. Gershwin received his medical degree from Stanford University in 1971. Pet'r's Ex. 98 at 1, ECF No. 38. He completed his internship and residency at Tufts-New England Medical Center in Boston, Massachusetts, before working as a clinical associate in immunology at the National Institutes of Health in Bethesda, Maryland. *Id.* at 2. He joined the University of California Davis School of Medicine faculty in 1975 as an assistant professor of rheumatology and allergy. *Id.* He has been a full-time professor since 1981 and was the chief of the medical school's division of rheumatology/allergy and clinical immunology for 38 years. *Id.* Dr. Gershwin is board certified by the American Board of Internal Medicine with a subspecialty in rheumatology and by the American Board of Allergy and Clinical Immunology. *Id.* Dr. Gershwin has received numerous honors, is a member of multiple professional societies, and has served as an editor of various journals. *Id.* at 3–8. He is an author or editor of 72 books or monographs and more than 1,000 other publications. *Id.* at 9–135.

2. Respondent's Expert, Dr. Arnold I. Levinson, M.D.

Dr. Levinson is an Emeritus Professor of Medicine and Neurology at the University of Pennsylvania, Perelman School of Medicine. Resp't's Ex. A at 1, ECF No. 47-1. He also holds the following positions: Chief of the Allergy and Immunology Section, Director of the Fellowship Training Program in Allergy and Immunology, Director of the Penn Center of Clinical Immunology, and Associate Dean for Research. *Id.* Dr. Levinson is board certified in internal medicine allergy and clinical immunology. *Id.* He is regarded as an expert in allergic and immunologic diseases. *Id.* For over 35 years, he maintained a clinical practice, evaluating and treating patients with a broad range of immune-mediated diseases, including autoimmune, hypersensitivity, and immunodeficiency disorders. *Id.* He also treated patients who experienced adverse reactions to drugs and vaccines. *Id.* Dr. Levinson has conducted research funded by the National Institutes of Health, the Veterans Administration, and several private foundations dealing with various aspects of autoimmunity, hypersensitivity, and immunodeficiency. *Id.*

3. Respondent's Expert, Maryanne Makredes Senna, M.D.

Dr. Senna is an Assistant Professor of Dermatology at Harvard Medical School and an attending board-certified dermatologist at Massachusetts General Hospital ("MGH") in Boston. Resp't's Ex. C at 1, ECF No. 47-3. She has clinical and research expertise in hair loss disorders, including AA. *Id.* She co-directs the MGH Hair Loss Clinic, where she sees 50+ new alopecia patient consults per month and receives national and international patient referrals. *Id.* At MGH, she directs the Hair Academic Innovative Research Unit, dedicated to hair loss disorders. *Id.* The unit conducts multiple ongoing clinical trials and epidemiologic studies for AA. *Id.* Her clinical trial research has been primarily focused on inflammatory hair loss disorders such as AA and lichen planopilaris. *Id.* at 2. Dr. Senna has published peer-reviewed articles on AA pathogenesis, associated conditions, and treatments, in addition to authoring a paper on how to properly diagnose hair loss conditions in pediatric patients. *Id.* at 1–2. She has also lectured and presented her alopecia research at national and international meetings, as listed in her curriculum vitae. *Id.*

4. Respondent's Expert, Emmanual Maverakis, M.D.

Dr. Maverakis is a member of the American Association of Immunologists. Resp't's Ex. H at 1, ECF No. 86-1. He holds medical board certifications in dermatology and clinical informatics. *Id.* He is a tenured full-time professor at the University of California, Davis, where he holds a variety of additional titles, including the Director of Autoimmunity and Director of Immune Monitoring. *Id.* He was a Howard Hughes Medical Institute fellow at the La Jolla Institute for Immunology, where he received additional training in immunology. *Id.* There, he studied molecular mimicry, epitope spreading, and T-cell antigen processing. *Id.* He has published 190 peer-reviewed, PubMed-indexed manuscripts. *Id.* at 2 As a clinician, he specializes in treating patients with rare diseases, including rare skin cancers and rare immune-mediated diseases. *Id.* He has been awarded visiting professorships/lectureships by a variety of institutions. *Id.*

B. Expert Reports

1. Petitioner's Expert, Dr. Gershwin

In his first expert report, Dr. Gershwin provided a general overview of alopecia. He explained that "[AA] consists of one or more circular bald patches around the scalp," while "[a]lopecia totalis includes complete loss of hair on the scalp and is the most extreme form of [AA]." Pet'r's Ex. 16 at 1, ECF No. 29. He noted that AA and alopecia totalis are "considered inflammatory and non-scarring" and that AA occurs in about 1.7% of people at some point during their lifetime. *Id.* at 1–2. Additionally, he claimed that "alopecia universalis is diagnosed with 100% loss of both scalp and body hair." *Id.* at 2. Dr. Gershwin further explained that alopecia is so dynamic that patches of hair loss "may be seen in other areas of the body such as elbow, arms, and thigh." *Id.* He continued that "the disease may involve facial hair, including eyelashes, eyebrows, and the beard area." *Id.* Dr. Gershwin also noted that alopecia is a systemic disease that can affect the nails and eyes in addition to the hair follicles. *Id.*

Dr. Gershwin then explained that "[t]he genetic basis of alopecia is strongly supported by its observed heritability in first degree relatives, twin studies[,] and genetic linkage analysis of alopecia families." *Id.* He noted that 10% to 42% of alopecia patients have a first-degree relative who is also affected. *Id.* Among these cases, "7% of patients have at least one parent with alopecia, 3% have at least one sibling with the disease, and a minority of 2% have a child who suffers from alopecia." *Id.* Citing Martinez-Mir et al.,[12] Dr. Gershwin asserted that "[a] genome wide search for linkage of 20 families with alopecia consisting of 102 affected and 118 unaffected individuals demonstrated the association of [human leukocyte antigens ("HLA")][13] with alopecia." *Id.* (citing Pet'r's Ex. 21, ECF No. 41-5). Dr. Gershwin identified multiple HLA class I and II alleles "that potentially contribute to the diagnosis of alopecia," and noted DQ3, DR11, and DQ7 are associated with the more severe iterations of the disease: alopecia totalis and universalis. *Id.* There were several other associations, which Dr. Gershwin discussed, of certain alleles found more commonly in individuals from specific geographical locations and ethnic backgrounds and in alopecia

[12] Amalia Martinez-Mir et al., *Genomewide Scan for Linkage Reveals Evidence of Several Susceptibility Loci for Alopecia Areata*, 80 AM. J. HUM. GENETICS 316 (2007).

[13] HLA are "histocompatibility antigens governed by genes of the HLA complex (the human major histocompatibility complex), a region on the short arm of chromosome [six] containing several genetic loci, each having multiple alleles." *Dorland's* at 103.

patients. *Id.* Citing Petukhova et al.,[14] Dr. Gershwin stated that in a genome-wide association study of 1,054 alopecia cases and 3,278 controls, it was "reported that eight genomic regions, including loci encoding genes in both innate and adaptive immunity [were] associated with alopecia." *Id.* (citing Pet'r's Ex. 26, ECF No. 41-10). He explained that the effectiveness of immunosuppressive agents in treating alopecia indicated that it is autoimmune and that HLA "has been reported to play a major role in the etiology of autoimmunity." *Id.* at 3 (citing Pet'r's Ex. 53, ECF No, 33-7;[15] Pet'r's Ex. 54, ECF No. 33-8).[16] As "confirmation of this specific hypothesis," Dr. Gershwin notes "the increased expression of specific HLAs in alopecia patients such as HLA-DR, HLA-A, HLA-B, and HLA-C, which are rarely seen in healthy individuals[,] as well as the identification of a number of genetic risk factors within various innate and adaptive immunity gene loci [ ]." *Id.* (citing Pet'r's Ex. 55, ECF No. 33-9[17] Pet'r's Ex. 56, ECF No. 33-10;[18] Pet'r's Ex. 26). Indeed, "[t]he most widely affected hypothesis for the effector mechanism of alopecia is the destruction of the [hair follicle], an immune privilege site." *Id.*

Dr. Gershwin explained that in addition to a genetic component, the autoimmune nature of alopecia is "strongly supported by clinical observations that patients with AA are often diagnosed with one or more other autoimmune disorders, including vitiligo, lupus erythematosus, myasthenia gravis, scleroderma, ulcerative colitis, Type I diabetes, thyroiditis, celiac disease, and rheumatoid arthritis." *Id.* (citing Pet'r's Ex. 47, ECF No. 33-1;[19] Pet'r's Ex. 51, ECF No. 33-5).[20] Additionally, stress hormones "are known to affect alopecia," and "exposure to ultraviolet light, natural and chemical bodily offenses, physical injury, and emotional distress" have been linked to alopecia. *Id.* (citing Pet'r's Ex. 35, ECF No. 31-9).[21] More generally, "[c]linical and experimental studies [have] show[n] that environmental insults such as emotional/physical stressors, hormones and infections contribute to autoimmunity," and autoimmune disease. *Id.* (citing Pet'r's Ex. 31, ECF No. 31-5;[22] Pet'r's Ex. 32, ECF No. 31-6;[23] Pet'r's Ex. 33, ECF No. 31-7;[24] Pet'r's Ex. 34, ECF

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[14] Lynn Petukhova et al., *Genome-Wide Association Study in Alopecia Areata Implicates Both Innate and Adaptive Immunity*, 466 NATURE 113 (2010).
[15] Nili Avidan et al., *Genetic Basis of Myasthenia Gravis - a Comprehensive Review*, 52 J. AUTOIMMUNITY 146 (2014).
[16] Alia Hasham et al., *Genetic Analysis of Interferon Induced Thyroiditis (IIT): Evidence for a Key Role for MHC and Apoptosis Related Genes and Pathways*, 44 J. AUTOIMMUNITY 61 (2013).
[17] D. Martin Carter & Brian V. Jegasothy BV, *Alopecia Areata and Down syndrome*, 112 ARCHIVES DERMATOLOGY 1397 (1976).
[18] Amos Gilhar et al., *Lymphocytes, Neuropeptides, and Genes Involved in Alopecia Areata*, 117 J. CLINICAL INVESTIGATION 2019 (2007).
[19] William V.R. Shellow, *Profile of Alopecia Areata: A Questionnaire Analysis of Patient and Family*, 31 INT'L J. DERMATOLOGY 186 (1992).
[20] S. Sipetić et al., *Family History and Risk of Type 1 Diabetes Mellitus*, 39 ACTA DIABETOLOGICA 111 (2002).
[21] Taisuke Ito, *Recent Advances in the Pathogenesis of Autoimmune Hair Loss Disease Alopecia Areata,* 2013 CLINICAL DEVELOPMENTAL IMMUNOLOGY 1 (2013).
[22] Carlo Perricone et al., *Autoimmune/Inflammatory Syndrome Induced by Adjuvants (ASIA) 2013: Unveiling the Pathogenic*, *Clinical and Diagnostic Aspects,* 47 J. AUTOIMMUNITY 1 (2013).
[23] Shiv Pillai, *Rethinking Mechanisms of Autoimmune Pathogenesis*, 45 J. AUTOIMMUNITY 97 (2013).
[24] Carlo Selmi et al., *Mechanisms of Environmental Influence on Human Autoimmunity: A National Institute of Environmental Health Sciences Expert Panel Workshop*, 39 J. AUTOIMMUNITY 272 (2012).

No. 31-8).[25] Dr. Gershwin stated that viruses, such as "hepatitis B, hepatitis C, Epstein-Barr, and swine flu [] have also been suggested to trigger alopecia []." *Id.* (citing Pet'r's Ex. 43, ECF No. 32-7;[26] Pet'r's Ex. 44, ECF No. 32-8;[27] Pet'r's Ex. 45, ECF No. 32-9;[28] Pet'r's Ex. 46, ECF No. 32-10;[29] Pet'r's Ex. 47). He stated that, "[t]heories have also been put forward regarding seasonal associations, with evidence of increased disease relapses between the months of February and March." *Id.* He further explained that "[t]his may also be a result of the high multitudes of viruses in early spring, supporting the hypothesis that alopecia may be an effect of certain viral infections." *Id.*

Dr. Gershwin asserted that individuals without alopecia maintain immune privilege within the hair follicle "in multiple ways, such as omitting [major histocompatibility complex ("MHC")][30] class I in the proximal outer root sheath [, while] patients identified with alopecia have a strong association with those same MHC class I alleles." *Id.* at 3–4. He explained that in alopecia patients, "[a]utoreactive cytotoxic T cells[31] target melanogenesis-associated peptides [in hair follicles] that produce melanin pigment." *Id.* at 4 (citing Pet'r's Ex. 57, ECF No. 34-1;[32] Pet'r's Ex. 58, ECF No. 34-2).[33] According to Dr. Gershwin, this process results in "the sparing of unpigmented hairs, and regrowth of initially white or gray hairs following the onset of alopecia." *Id.* (citing Pet'r's Ex. 23, ECF No. 41-7).[34]

Dr. Gershwin also asserted that cytokines[35] and natural killer ("NK") cells[36] have been implicated in the breakdown of immune privilege in alopecia. *Id.* at 3–4. For example, NKG2D

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[25] Karen H. Costenbader et al., *Genes, Epigenetic Regulation and Environmental Factors: Which is the Most Relevant in Developing Autoimmune Diseases,* 11 AUTOIMMUNITY REVS. 604 (2011).

[26] Nesrine Gamal et al*., Alopecia Universalis After Discontinuation of Pegylated Interferon and Ribavirin Combination Therapy for Hepatitis C: A Case Report,* 13 ANNALS HEPATOLOGY 293 (2014).

[27] David A. Geier & Mark R. Geier*, A Case-Control Study of Serious Autoimmune Adverse Events Following Hepatitis B Immunization*, 38 AUTOIMMUNITY 295 (2005).

[28] Taisuke Ito, *Advances in the Management of Alopecia Areata,* 39 J. OF DERMATOLOGY, (2012).

[29] Thomas A. Rodriguez et al., *Onset of Alopecia Areata after Epstein-Barr Virus Infectious Mononucleosis*, 59 J. AM. ACAD. DERMATOLOGY 11 (2008).

[30] MHC are "the genes determining the major histocompatibility antigens, in all species a group of closely linked multiallelic genes located in a small region on one chromosome." *Dorland's* at 391.

[31] Cytotoxic T cells are "differentiated T lymphocytes that can recognize and lyse target cells bearing specific antigens recognized by their antigen receptors." *Dorland's* at 1070. Cytotoxic T cells are a type of CD8 T cell, which is a T cell that "carr[ies] the CD8 antigen[.]" *Id.* at 311.

[32] Ralf Paus et al., *Is Alopecia Areata an Autoimmune-Response Against Melanogenesis-Related Proteins, Exposed by Abnormal MHC Class I Expression in the Anagen Hair Bulb,* 66 YALE J. BIOLOGY MED. 541 (1994).

[33] Amos Gilhar et al., *Melanocyte-Associated T Cell Epitopes can Function as Autoantigens for Transfer of Alopecia Areata to Human Scalp Explants on Prkdc$^{(scid)}$ Mice*, 117 J. INVESTIGATIVE DERMATOLOGY 1357 (2001).

[34]Abdullateef A. Alzolibani, *Epidemiologic and Genetic Characteristics of Alopecia Areata (Part 1)*, 20 ACTA DERMATOVENEROLOGICA ALPINA, PANNONICA, ET ADRIATICA 191 (2011).

[35] Cytokine is "a generic term for nonantibody proteins released by one cell population (e.g., primed T lymphocytes) on contact with a specific antigen, which act as intercellular mediators, as in the generation of an immune response." *Dorland's* at 460.

[36] NK cells are "cells capable of mediating cytotoxic reactions without prior sensitization against the target." *Dorland's* at 316.

NK cells "attack by recognizing certain glycoproteins, CMV UL16 proteins, and MHC class I related proteins MlCA/MICB." *Id.* (citing Pet'r's Ex. 74, ECF No. 35-8).[37] Further, "[i]n alopecia patients, the outer root sheath presents MICA proteins, leading NKG2D + NK cells to target the HF." *Id.* (citing Pet'r's Ex. 60, ECF No. 34-4).[38] He explained that the process causes "[t]he breaking of tolerance in HF and subsequent change in cytokine/chemokine profiles lead[ing] to infiltration of lymphocytes." *Id.* (citing Pet'r's Ex. 26; Pet'r's Ex. 35).

Dr. Gershwin further asserted that specific "[c]hemokines [that] are involved with the development of autoimmune interactions[,]are more prominent in alopecia patients than healthy individuals." *Id.* (citing Pet'r's Ex. 63, ECF No. 34-7;[39] Pet'r's Ex. 64, ECF No. 34-8;[40] Pet'r's Ex. 65, ECF No. 34-9;[41] Pet'r's Ex. 66, ECF No. 34-10;[42] Pet'r's Ex. 67, ECF No. 35-1).[43] To support this contention, Dr. Gershwin cited the C3H/HeJ spontaneous mouse model of alopecia, "which manifests the clinical pathological features of human alopecia, including infiltration of CD8 + NKG2D + T cells [from] around the epithelial layers of [hair follicles]." *Id.* at 4 (citing Pet'r's Ex. 75, ECF No. 35-9;[44] Pet'r's Ex. 76, ECF No. 35-10).[45] All of these different components, he asserted, defined "[t]he mechanism that leads to alopecia[,] includ[ing] genetic susceptibility and the generation of cytotoxic CD8 T cells." *Id.* at 5. He noted that "[i]mportantly, the signals required to generate CD8 T cells beyond the initial exposure are not required for proliferation[,] and indeed there is likely to be early immunological programming of such cytotoxic T cell expansion[]." *Id.* (citing Pet'r's Ex. 77, ECF No. 36-1;[46] Pet'r's Ex. 78, ECF No. 36-2;[47] Pet'r's Ex. 79, ECF No. 36-3;[48] Pet'r's Ex. 80, ECF No. 36-4;[49] Pet'r's Ex. 81, ECF No.

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[37] Stefan Bauer et al., *Activation of NK Cells and T cells by NKG2D, a Receptor for Stress-Inducible MICA*, 285 SCI. 727 (1999).
[38] Taisuke Ito et al., *Maintenance of Hair Follicle Immune Privilege is Linked to Prevention of NK Cell Attack*, 128 J. INVESTIGATIVE DERMATOLOGY 1196 (2008).
[39] Alessandro Antonelli et al., *Chemokine (C-X-C motif) Ligand (CXCL)10 in Autoimmune Diseases*, 13 AUTOIMMUNITY REVS. 272 (2014).
[40] Chiara Cordiglieri et al., *Innate Immunity in Myasthenia Gravis Thymus: Pathogenic Effects of Toll-Like Receptor 4 Signaling on Autoimmunity*, 52 J. AUTOIMMUNITY 74 (2014).
[41] Perrine Cufi et al., *Central Role of Interferon-Beta in Thymic Events Leading to Myasthenia Gravis*, 52 J. AUTOIMMUNITY 44 (2014).
[42] Eun Young Lee et al*., The Interaction Between CXCL10 and Cytokines in Chronic Inflammatory Arthritis*, 12 AUTOIMMUNITY REVS. 554 (2013).
[43] Sandrine Benoit et al., *Selective Expression of Chemokine Monokine Induced by Interferon-Gamma in Alopecia Areata*, 121 J. INVESTIGATIVE DERMATOLOGY 933 (2003).
[44] John P. Sundberg et al., *Alopecia Areata in Aging C3H/HeJ Mice*, 102 J. INVESTIGATIVE DERMATOLOGY 847 (1994).
[45] Luzhou Xing et al., *Alopecia Areata is Driven by Cytotoxic T Lymphocytes and is Reversed by JAK Inhibition*, 20 NATURE MED. 1043 (2014).
[46] Hannah Rabenstein et al., *Differential Kinetics of Antigen Dependency of CD4$^+$ and CD8$^+$ T Cells*, 192 J. IMMUNOLOGY 3507 (2014).
[47] Julius C. R. Hafalla et al., *Short-Term Antigen Presentation and Single Clonal Burst Limit the Magnitude of the CD8+ T Cell Responses to Malaria Liver Stages*, 99 PROC. NAT'L ACAD. SCIS. U.S. 1819 (2002).
[48] Susan M. Kaech & Rafi Ahmed, *Memory CD8$^+$ T Cell Differentiation: Initial Antigen Encounter Triggers a Developmental Program in Naïve Cells*, 2 NATURE IMMUNOLOGY 415 (2001).
[49] Roberto Mercado et al., *Early Programming of T Cell Populations Responding to Bacterial Infection*, 165 J. IMMUNOLOGY 6833 (2000).

36-5;[50] Pet'r's Ex. 82, ECF No. 36-6;[51] Pet'r's Ex. 83, ECF No. 36-7;[52] Pet'r's Ex. 84, ECF No. 36-8;[53] Pet'r's Ex. 85, ECF No. 36-9;[54] Pet'r's Ex. 86, ECF No. 36-10).[55] He further noted that "[o]nset within 14 days would certainly be consistent with generation of a CD8 response." *Id.*

Summarizing his medical theory, Dr. Gershwin wrote that Petitioner "developed alopecia because of his unique genetic susceptibility." *Id.* He claimed that Petitioner "received a vaccine that produced cytotoxic T cells that were directed at his hair follicles and [] cross react[ed] with an epitope or region of the Gardasil vaccine." *Id.* Dr. Gershwin supported this contention by mentioning that "[t]he kinetics and the production of such cytotoxic T cells is consistent and biologically plausible with the literature on the production of such cytotoxic T cells." *Id.* He explained that the mechanism is similar to the mouse model and "is due to molecular mimicry." *Id.*

In order to link the Gardasil HPV vaccine to the autoimmune attack on Petitioner's hair follicles, Dr. Gershwin cited Wise et al.[56] *Id.* (citing Pet'r's Ex. 88 at 3, ECF No. 37-2). He wrote that the data from Wise et al. "suggest[s] that HPV is the most likely initiating immunogen herein." *Id.* The authors noted their "speculat[ion] that cell growth cycles might be pathologically modulated by vaccine-induced antibodies." Pet'r's Ex. 88 at 3. The article "suggest[ed] antigenic similarities between vaccines and hair follicles, at least in susceptible patients, that should be investigated." *Id.* Alternatively, the authors conceded that "[u]nexpected hair loss could occasionally follow vaccine exposure by chance alone since vaccine exposures are extremely common, and unexplained hair loss ([AA] or other syndromes) is not rare." *Id.* Furthermore, they claimed that "possible biases in case ascertainment" was identified due to the gender and occupation of the study group. *Id.* Thus, the authors concluded that "individuals may be more likely to suspect recent immunization when they develop hair loss, and they are familiar with the importance of reporting adverse events." *Id.*

Next, Dr. Gershwin made some additional points noting that the variation in the human immune system is largely driven by non-heritable influences. Pet'r's Ex. 16 at 5–6. He further

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[50] Marianne J.B. van Stipdonk et al., *Dynamic Programming of CD8$^+$ T Lymphocyte Responses*, 4 NATURE IMMUNOLOGY 361 (2003).

[51] Matthew A. Williams & Michael J. Bevan, *Shortening the Infectious Period Does Not Alter Expansion of CD8 T Cells but Diminishes Their Capacity to Differentiate into Memory Cells*, 173 J. IMMUNOLOGY 6694 (2004).

[52] Phillip Wong & Eric G. Pamer, *Disparate in Vitro and in Vivo Requirements for IL-2 During Antigen-Independent CD8 T Cell Expansion*, 172 J. IMMUNOLOGY 2171 (2004).

[53] Gabriel R. Starbeck-Miller et al., *IL-12 and Type I Interferon Prolong the Division of Activated CD8 T Cells by Maintaining High-Affinity IL-2 Signaling in Vivo*, 211 J. EXPERIMENTAL MED. 105 (2014).

[54] Michael J. Bevan & Pamela J. Fink, *The CD8 Response on Autopilot,* 2 NATURE IMMUNOLOGY 381 (2001).

[55] David Masopust et al., *The Role of Programming in Memory T-Cell Development*, 16 CURRENT OP. IMMUNOLOGY 217 (2004).

[56] Robert P. Wise et al., *Hair Loss After Routine Immunizations*, 278 JAMA 1176 (1997). Wise et al. discussed 60 reports of hair loss following various vaccinations, but primarily hepatitis B vaccinations, identified through the Vaccine Adverse Event Reporting System ("VAERS"). After reviewing the 60 case reports, the authors noted that "immunizations warrant consideration among potential causes of hair loss. *Id.* at 3.

stated that "[r]ecent advances in technology now allow much more comprehensive surveys to be conducted across the many different components of the immune system." *Id.* at 6. He reasoned that "the immune system of healthy individuals is very much shaped by the environment and most likely by the many different microbes that an individual encounters in their lifetime." *Id.* He then concluded that "the immune response cannot always be predicted and that in fact rare events can be explained by stochastic events that have occurred in an individual's lifetime." *Id.* In any particular case, "for diseases as uncommon as [AA], one cannot rely on epidemiology because the power calculations are insufficient to provide the necessary data to draw statistical conclusions." *Id.*

In a responsive report, Dr. Gershwin explained that he could not identify the specific component of the Gardasil vaccine that triggered the cytotoxic T cell response. *See generally* Pet'r's. Ex. 99, ECF No. 64. He asserted that "identification and definition of epitopes is extraordinarily complex and requires data that is not available and could not be achieved without a **major** research effort." *Id.* at 1 (emphasis in original). Dr. Gershwin referred to the Colmenares et al.[57] article to note "the imbalance of CD8 T cells following Gardasil vaccine[.]" *Id.* at 2 (citing Pet'r's Ex. 105, ECF No. 65-6). He then quoted the authors' observations,

> Interestingly, [the authors] observed that while the proportion of CD8+ lymphocytes that expressed ILT2 decreased after immunization, there was an increase in the density (MFI) of this receptor in these cells. Although the underlying mechanism for this apparent paradoxical effect remains to be determined, [their] data suggest[ed] that in the fraction of CD8+ cells that remain ILT2 positive after quadrivalent HPV (type 6/11/16/18) vaccine immunization, the enhanced density of this receptor may exert an increased regulatory effect[.]

Id. (quoting Pet'r's Ex. 105 at 7).

In a supplemental report, Dr. Gershwin asserted that Gardasil contains two components that could affect the production of CD8+ T cells. Pet'r's Ex. 106 at 1–2, ECF No. 68. He acknowledged literature that notes the addition of an aluminum adjuvant to Gardasil "is limited by weak stimulation of cell-mediated immunity, [and] this can be enhanced by the addition of other immunomodulatory molecules." *Id.* at 1 (quoting Pet'r's Ex. 107 at 1, ECF No. 69-1).[58] Hogenesch endeavored to "increase our understanding of the mechanisms involved in adsorption of antigens onto aluminum adjuvants and the effect of adsorption on the stability of antigens and the immune response." Pet'r's Ex. 107 at 9. The article abstract noted that aluminum adjuvants "support[ed] activation of CD8 T cells, but these cells d[id] not undergo terminal differentiation to cytotoxic T cells." *Id.* at 1. The author further noted that "[a]luminum adjuvants primarily enhance antibody production and have little effect on the cell-mediated arm of the immune response." *Id.* at 2.

Dr. Gershwin stated that the Gardasil vaccine contained polysorbate 80, which is "added to vaccines as an emulsifier to spread the antigenic components of the vaccine and facilitate the

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[57] V. Colmenares et al., *Human Papillomavirus Immunization is Associated with Increased Expression of Different Innate Immune Regulatory Receptors,* 19 CLINICAL VACCINE IMMUNOLOGY 1005 (2012).
[58] Harm Hogenesch, *Mechanism of Immunopotentiation and Safety of Aluminum Adjuvants*, 3 FRONTIERS IMMUNOLOGY 1 (2013).

production of cell-mediated immunity." Pet'r's Ex. 106 at 1. He cites Macleod et al.,[59] to assert that "[t]his point [was] studied specifically with respect to CD8 cells in literature that describes the mechanism" *Id.* (Pet'r's Ex. 108, ECF No. 69-2). Dr. Gershwin explained that "[t]he use of other materials in vaccines, when alum is included, to facilitate cell-mediated immunity has been known for decades." *Id.* at 2 (citing Pet'r's Ex. 112, ECF No. 69-6).[60] He opined that "there is literature that does document T cell immune responses, measured as inflammatory cytokine production, of peripheral blood cells in young women immunized with HPV." *Id.* at 2 (citing Pet'r's Ex. 111, ECF No. 69-5).[61] The literature cited by Dr. Gershwin is the Toh et al. study, whose authors sought "to document the elevated cellular immune responses in girls who were previously vaccinated with at least [one] dose of [quadrivariate HPV] when compared with unvaccinated girls, [six] years following the last dose." Pet'r's Ex. 111 at 8.

Additionally, he reiterated that "the mechanism herein is molecular mimicry at the T cell level [and restated that Petitioner] would not have developed alopecia if he did not uniquely have an antigen in his hair follicles that was cross-reactive with a component of the HPV vaccine." Pet'r's Ex. 106 at 2. He further stated that Petitioner did not have "an abnormal initial immune response to the Gardasil, rather that the pathology is due to molecular mimicry via the cross reactive antigen." *Id.* He opined that Petitioner's "self-antigen is always present and is the basis for the continued production of cytotoxic CD8 T cells and subsequent alopecia." *Id.* In support of this argument, Dr. Gershwin cited Kaech et al., which showed that naïve CD8+ T cells differentiate in effector and memory cells following antigenic stimulation. Pet'r's Ex. 113 at 1. The article described how CD8+ T cells become activated and differentiate into effector cytotoxic T lymphocytes, following exposure to an antigen such as an infection or vaccine. *Id.* The authors "propose[d] that the most effective T cell vaccines will be those that produce the optimal burst of antigen expression and recruit the greatest number of antigen-specific CD8+ T cells into the response." *Id.* at 7. Two additional articles, Cho et al.[62] and Veiga-Fernandes et al.,[63] further discussed the effect that antigen stimulation has on naïve CD8+ cells. *See* Pet'r's Ex. 114, ECF No. 69-8; Pet'r's Ex. 115, ECF No. 69-9). Dr. Gershwin reiterated that he could not provide the sequence or identification of hair follicle antigen but asserted that molecular mimicry is well accepted and the identification of such antigenic determinants would require a major research effort. Pet'r's Ex. 106 at 2.

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[59] Megan K.L. MacLeod et al., *Vaccine adjuvants aluminum and monophosphoryl lipid A provide distinct signals to generate protective cytotoxic memory CD8 T cells,* 108 PROCS. NAT'L ACAD. SCIS. U.S. 7914 (2011).

[60] Noelene E. Byars & Anthony C. Allison, *Adjuvant Formulation for Use in Vaccines to Elicit Both Cell-Mediated and Humoral Immunity*, 5 VACCINE 223 (1987).

[61] Zheng Quan Toh et al., *Cellular Immune Responses 6 Years Following 1, 2, or 3 Doses of Quadrivalent HPV Vaccine in Fijian Girls and Subsequent Responses to a Dose of Bivalent HPV Vaccine*, 5 OPEN F. INFECTIOUS DISEASES 1 (2018).

[62] Bryan K. Cho et al., *Functional Differences Between Memory and Naive CD8 T Cells*, 96 PROC. NAT'L ACAD. SCI. U.S. 2976 (1999).

[63] Henrique Veiga-Fernandes et al., *Response of Naïve and Memory CD8$^+$ T Cells to Antigen Stimulation in Vivo,* 1 NATURE IMMUNOLOGY 47 (2000).

In Dr. Gershwin's final submitted report, he briefly reiterated "the importance (and difficulty) of epitope production." Pet'r's Ex. 116 at 1 (citing Pet'r's Ex. 119, ECF No. 78-3;[64] Pet'r's Ex. 120, ECF No. 78-4;[65] Pet'r's Ex. 121, ECF No. 78-5).[66] He cited literature in support of this assertion as well as "recent literature of an increased incidence of [AA] after HPV infection." *Id.* (citing Pet'r's Ex. 118, ECF No. 78-2).[67] Tu et al. summarized an "investigat[ion of] the correlation between a history of [HPV] infection and [AA] risk." Pet'r's Ex. 118 at 1. The authors identified "30,001 patients with newly diagnosed HPV infection between 2000 and 2012." *Id.* Patients who did not have HPV, "were randomly selected as the comparison cohort. HPV infection cohort were matched to comparison individuals at a 1:1 ratio by age, gender and index year." *Id.* The authors ultimately concluded that "[a] history of HPV infection is associated with the development of subsequent [AA] in Taiwanese subjects." *Id.*

2. Respondent's Expert, Dr. Levinson

Dr. Levinson concurred with Petitioner's "initial diagnosis of [AA] and later diagnosis of alopecia universalis." Resp't's Ex. A at 3; ECF No. 47-1. However, he disagreed with the "assignment of causality" to the vaccine Petitioner received on March 30, 2015, or any prior Gardasil vaccination that Petitioner received. *Id.*

According to Dr. Levinson, "local injection site reactions including pain, erythema and swelling were more common after vaccine than placebo but systemic adverse effects including development of systemic disease and presumed autoimmune disease were not." *Id*. at 3.

Dr. Levinson cited a study by Chao et al.,[68] which evaluated 189,629 women of all ages who received at least one dose of the vaccine and another group of women who did not receive the vaccine to compare the incidence of autoimmune disease among the two groups. *Id.* (citing Resp't's Ex. C, Tab 58, at 2, ECF No. 62-8). Ultimately, this study showed "no clear evidence of safety signal for [autoimmune] conditions following vaccination with HPV4[.]" Resp't's Ex. C, Tab 58, at 9. Dr. Levinson also cited Arnheim-Dahlström et al.[69] from 2013 which evaluated 997,585 girls aged 10-17 years, 296,826 of whom had received quadrivalent HPV vaccine. *Id.* at 4 (citing Resp't's Ex. C, Tab 56, ECF No. 62-6). Within this study, the investigators "identified no safety signals with respect to autoimmune, neurological, and venous thromboembolic events after the HPV vaccine had been administered." Resp't's Ex. C, Tab 56, at 5. Dr. Levinson cited

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[64] Mette Voldby Larsen et al., *An Integrative Approach to CTL Epitope Prediction: A Combined Algorithm Integrating MHC Class I Binding, TAP Transport Efficiency, and Proteasomal Cleavage Predictions*, 35 EUR. J. IMMUNOLOGY 2295 (2005).

[65] Claus Lundegaard et al., *State of the Art and Challenges in Sequence Based T-Cell Epitope Prediction*, 6 IMMUNOME RSCH. 1 (2010).

[66] Ruth E. Soria-Guerra et al., *An Overview of Bioinformatics Tools for Epitope Prediction: Implications on Vaccine Development*, 53 J. BIOMEDICAL INFORMATICS 405 (2015).

[67] Ting-Yu Tu et al., *Human Papillomavirus Symptomatic Infection Associated with Increased Risk of New-Onset Alopecia Areata: A Nationwide Population-Based Cohort Study*, 119 J. AUTOIMMUNITY (2021).

[68] C. Chao et al., *Surveillance of Autoimmune Conditions Following Routine use of Quadrivalent Human Papillomavirus Vaccine*, 271 J. INTERNAL MED. 193 (2011).

[69] Lisen Arnheim-Dahlström, *Autoimmune, Neurological, and Venous Thromboembolic Adverse Events After Immunisation of Adolescent Girls with Quadrivalent Human Papillomavirus Vaccine in Denmark and Sweden: Cohort Study*, 347 BMJ (2013).

three additional studies that found no evidence of an increased risk for autoimmune disorders following HPV vaccinations in large populations of girls. Resp't's Ex. A at 3–4; *see* Resp't's Ex. A, Tab 4, ECF No. 70-4;[70] Resp't's Ex A, Tab 5, ECF No. 70-5;[71] Resp't's Ex. A, Tab 6, ECF No. 70-6;[72] Resp't's Ex. A, Tab 7, ECF No. 70-7.[73]

Describing alopecia spectral disorders as "products of autoimmune mechanisms that injure hair follicles," Dr. Levinson again agreed with Dr. Gershwin that "[e]pidemiologic studies clearly demonstrate an underlying genetic basis to these disorders as well as a number of environmental and emotional/physical stress factors." *Id.* at 4. He noted that "cytotoxic CD8+ T cells and NK cells are thought to target autoantigens expressed on hair follicles." *Id.* Further, he stated that "[d]ata also suggest that CD4+ T cells and a myriad of soluble factors, e.g., cytokines and chemokines, contribute to injury of the hair follicle." *Id.*

Dr. Levinson's disagreement with Dr. Gershwin lies in the "giant leap of faith" necessary to invoke molecular mimicry in connecting the HPV vaccine to the development of alopecia. *Id.* at 5. Dr. Levinson explained that Dr. Gershwin "failed to identify any semblance of molecular mimicry between molecular motifs expressed on the hair follicle and the viral antigens that are found in the Gardasil vaccine." *Id.* According to Dr. Levinson, accepting Dr. Gershwin's opinion would "legitimize the claim that literally any exogenous antigen, be it a vaccine component, an infectious agent, etc., can cause just about any autoimmune disease by the process of molecular mimicry despite there being no demonstrable evidence for said molecular mimicry between the putative exogenous agent and the relevant target self-antigen." *Id.*

Dr. Levinson then stated that "there is no reliable epidemiological evidence that supports the proposition that the Gardasil vaccine causes [AA]." *Id.* Dr. Levinson stated that Dr. Gershwin mischaracterized the Wise et al. findings. *Id.* (citing Pet'r's Ex. 88 at 3). According to Dr. Levinson, "[t]here was no mention of whether the alopecia was characterized by clinical features of [AA], and the onset of alopecia occurred at variable times after vaccination." *Id.* He asserted that "most of these incidents occurred within [one] month of immunization with a variety of vaccines, and some occurred within one day of vaccination." *Id.* Additionally, Dr. Levinson noted that "patients ranged in age from neonates to 84 years-old. [Furthermore, s]everal of the patients experienced complete regrowth of hair after an episode of alopecia." *Id.* at 5–6. Notably, Dr. Levinson stated "that none of the subjects had received Gardasil, or for that matter any HPV vaccine" *Id.* at 6. Dr. Levinson then identified a case study of the development of AA after Gardasil vaccination involved a "[two-]year-old Japanese boy who developed alopecia areata/totalis one week and [three] days after receiving a third dose of Japanese encephalomyelitis vaccine and third dose of influenza vaccine, respectively." *Id.* (citing Resp't's Ex. A, Tab 14, ECF No. 71-7).[74]

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[70] L. Grimaldi-Bensouda et al., *Autoimmune Disorders and Quadrivalent Human Papillomavirus Vaccination of Young Female Subjects*, 275 J. INTERNAL MED. 398 (2014).
[71] Sara Miranda et al., *Human Papillomavirus Vaccination and Risk of Autoimmune Diseases: A Large Cohort Study of over 2 Million Young Girls in France*, 35 VACCINE 4761 (2017).
[72] Lamiae Grimaldi-Bensouda et al., *Risk of Autoimmune Diseases and Human Papilloma Virus (HPV) Vaccines: Six Years of Case-Referent Surveillance*, 79 J. AUTOIMMUNITY 84 (2017).
[73] Erin Y. Liu et al., *Quadrivalent Human Papillomavirus Vaccination in Girls and the Risk of Autoimmune Disorders: The Ontario Grade 8 HPV Vaccine Cohort Study*, 190 CANADIAN MED. ASS'N J. 648 (2018).
[74] Chien-Ho Chu et al., *Alopecia Areata After Vaccination: Recurrence with Rechallenge*, 33 PEDIATRIC DERMATOLOGY 218 (2016).

Dr. Levinson clarified his objection to Dr. Gershwin's causality theory in his supplemental report. Resp't's Ex. E, ECF No. 63-1. He explained that Dr. Gershwin's opinion has "no evidence of molecular mimicry between viral antigens in the Gardasil vaccine and self antigens expressed on hair follicles." *Id.* at 1. He further stated that there is no "scientific literature that supports the contention that any type of molecular mimicry accounts for the autoimmune destruction of hair cells in AA" *Id.*

Dr. Levinson responded to Dr. Gershwin's claim that "cytotoxic CD8+ T cells were key contributors to the destruction of hair follicles." *Id.* at 2–3. He criticized this view as flawed, stating that it is unlikely Gardasil would have triggered the development of any cytotoxic CD8+ T cells *Id.* at 3.

Furthermore, Dr. Levinson explained that "Gardasil, like so many commercial vaccines administered in the U.S., contains an adjuvant, namely an aluminum compound that potentiates the induced specific immune response." *Id.* And, he noted, "the principle immune response elicited by an aluminum compound-adjuvanted vaccine is essentially an antibody response." *Id.* (citing Resp't's Ex. E, Tab 5, ECF No. 63-6). He continued, "vaccines adjuvanted with aluminum salts, are very effective generating potent antibody responses but are ineffective generating protective cell-mediated immune responses, especially cytotoxic T cell responses." *Id.* (citing Resp't's Ex. E, Tab 5). Dr. Levinson noted that "[t]his limitation is the overriding impetus for developing vaccines formulated with novel adjuvants that elicit more protective broad-based immune responses, including cytotoxic T cell response, when administered in humans." *Id.* Thus, he concluded that it was "extremely unlikely that Gardasil induced the development of cytotoxic CD8+ T cells, let alone ones that are specific for any epitopes that are putatively shared by Gardasil viral antigens and self-antigens expressed on hair follicles." *Id.*

Dr. Levinson also noted that he "was unable to find a single case report that linked the wild-type infection with HPV to the development of AA." *Id.* Dr. Levinson explained that although identification of T cell epitopes and molecular mimicry is a complex subject, "it is certainly possible to identify autoimmune diseases in which mimicry of exogenous antigens by self antigens is pathogenic." Resp't's Ex. F at 2, ECF No. 66-1. He further stated that the difficulty of generating data to support an expert's causality opinion is immaterial because it would "undermine the merits of the claim being made." *Id.* Dr. Levinson then went on to note that he does not consider the absence of epidemiologic evidence when constructing his opinion during vaccine injury cases. *Id.* Instead, he relied on the totality of the experiential data in formulating his opinion. *Id.* Dr. Levinson then stated that he is more compelled to question a vaccine's causality when there are only a few published case reports that possibly link a particular vaccine to a disease. *Id.*

Clarifying his statement that it was unlikely that Gardasil would have induced any type of cytotoxic T cells, Dr. Levinson noted that he was "referring to cytotoxic CD8+ T cells that were in this case alleged to actively destroy hair follicles." *Id.* at 3. He further stated, "that such an outcome following Gardasil vaccination would be extremely unlikely even if molecular mimicry was operative, since this alum-adjuvanted vaccine, like all other alum-adjuvanted vaccines, do not induce the emergence of functional CD8+ T cells, i.e., cells that kill their targets in response to specific vaccine antigens." *Id.* He stated that Dr. Gershwin's reliance on the Colmenares et al. publication was flawed. *Id.* (citing Pet'r's Ex. 99 at 2). He noted that the authors "analyzed the

expression and function of Immunoglobulin-like transcript 2 (ILT2) as well as the expression of other NK cell receptors, which seem to participate in the innate immune response against different viruses." *Id.* at 3 (citing Pet'r's Ex. 105). Dr. Levinson acknowledged the imbalance identified by Dr. Gershwin but also pointed out that, in summarizing their results, the authors stated, "it is also possible that [the induction of ILT2 expression by quadrivalent HPV] is mediated by cytokines released by T cells." *Id.* (quoting Pet'r's Ex. 105 at 7). He argued that, in context, the study only showed that the authors observed a change in the expression of ILT2 on CD8+ T cells following vaccination. *Id.* Thus, he asserted that this did not provide any "demonstra[tion] that the Gardasil vaccination caused the development of any type of functional CD8+ cytotoxic T cells." *Id.* They concluded "that "HPV immunization is associated with significant changes in the expression and function of different innate immune receptors, including ILT2, which may participate in the protective effect of HPV vaccines." *Id.* (quoting Pet'r's Ex. 105 at 1). Dr. Levinson opined that "this paper clearly did not demonstrate that Gardasil vaccination caused the development of any type of functional CD8+ cytotoxic T cells" and that "no evidence has been presented to critique his theory." *Id.*

In a supplemental report, Dr. Levinson addressed Dr. Gershwin's idea about polysorbate 80 and how it elicits a stronger immune response. Resp't's Ex. G, ECF No. 75-1. He opined that Dr. Gershwin's discussion of this issue is irrelevant and fails to prove that Gardasil facilitates active CD8+ cytotoxic T cells. *Id.* at 1–2. Dr. Levinson then pointed to a previously cited study, Macleod et al., to argue that it reinforced his "assertion that vaccines adjuvanted only with an aluminum salt do not induce the development of antigen-specific functionally active cytotoxic CD8+ T cells." *Id.* at 2 (citing Pet'r's Ex. 108). However, he explained, "when a vaccine containing alum is 'fortified' with certain other immunomodulators like MPLA, the vaccine gains the capacity to induce the cytotoxic CD8+ T cells." *Id.* He goes on to state that Dr. Gershwin failed to mention that "polysorbate 80 and any of the other ingredients in the Gardasil vaccine do not possess such alum-fortifying adjuvant action." *Id.* Dr. Levinson reiterated that no literature supports Dr. Gershwin's statements, and asserted that instead, Dr. Gershwin has "conflated T cell-induced cytokine responses with T cell-induced cytotoxic T cell responses." *Id.*

3. Respondent's Expert, Dr. Senna

Dr. Senna's expert report began with a summary of Petitioner's medical history, his clinical presentation, and ultimate alopecia diagnosis. Resp't's Ex. C at 2–6, ECF No. 47-3 She then defined AA and described it as "an autoimmune disorder characterized by patches of non-scarring hair loss that can affect the scalp and/or the body." *Id.* at 6. Dr. Senna explained that her process of evaluating AA patients includes reviewing a patient's "extended history of the hair loss and other past medical history, family history, medications, and potential exacerbating factors." *Id.* at 10. She then looks for "clues that are supported by medical evidence that more than likely affect an individual's disease course, response to treatment, and overall prognosis." *Id.* Dr. Senna stated that she applied the same process to Petitioner's case and concluded that the HPV vaccine did not play a role in causation. *Id.*

Dr. Senna noted that AA affects up to 3% of the general population. *Id.* Like many autoimmune disorders, AA commonly presents and peaks during adolescence and early childhood.

Id. (citing Resp't's Ex. C, Tab 54, ECF No. 62-4).[75] Dr. Senna explained that this is the time when individuals receive the HPV vaccine series, which would lead one to "attribute the development of autoimmune conditions to the vaccine." *Id.* at 10–11. However, "large epidemiologic studies have not detected any such associations." *Id.* at 11 (citing Resp't's Ex. C, Tab 55, ECF No. 62-5;[76] Resp't's Ex. C, Tab 56, ECF No. 62-6;[77] Resp't's Ex. C Tab 57, ECF No. 62-7;[78] Resp't's Ex. C, Tab 58, ECF No. 62-8;[79] Resp't's Ex. C, Tab 59, ECF No. 62-9).[80] Dr. Senna cited a paper from Genovese et al.[81], detailing "[a] large meta-analysis on six studies on bivalent and quadrivalent HPV vaccines that in 243,289 patients who received the HPV vaccines [revealed] no correlation between autoimmune disorders and HPV vaccination." *Id.* (citing Resp't's Ex. C, Tab 50, ECF No. 61-10). Further, Skufca et al.[82] studied "134,615 11- to 15-year-olds vaccinated with the HPV vaccine [which] showed that vaccination was not associated with a higher risk of any adverse outcome during the entire [three] year follow-up." *Id.* (citing Resp't's Ex. C, Tab 51, ECF No. 62-1). Dr. Senna noted that a review of VAERS revealed 60,461,220 females and males who received HPV vaccines between 2009 and 2015 in the United States. *Id.* In these submitted cases, "dizziness and syncope were the most commonly reported nonserious [adverse events] in both males and females." *Id.* Dr. Senna further noted that this study showed "no reports of [AA] or other forms of hair loss." *Id.* Dr. Senna asserted that, in fact, "[t]here is no evidence that HPV vaccination causes [AA] or other autoimmune diseases." *Id.* at 11.

In Petitioner's case, Dr. Senna stated that Petitioner "had a number of baseline poor prognostic risk factors, including a history of atopy (allergic rhinitis, seasonal allergies), development at a young age (age 13)." *Id.* at 12. In Dr. Senna's expert opinion, "any one of the above [three] factors on its own would portend a poorer AA prognosis for a patient, making it more likely than not that the patient would be resistant to treatment, have a progressive course, and develop severe [AA] over time." *Id.* at 12–13.

Dr. Senna noted an that, "[e]xogenous stress[,] has been implicated as a trigger for AA exacerbations." *Id.* at 13 (citing Resp't's Ex. C, Tab 35, ECF No. 60-5;[83] Resp't's Ex. C, Tab 36,

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[75] S. Macleod & R.E. Appleton, *Neurological Disorders Presenting Mainly in Adolescence*, 92 ARCHIVES DISEASE CHILDHOOD 170 (2007).

[76] Julianne Gee et al., *Monitoring the Safety of Quadrivalent Human Papillomavirus Vaccine: Findings from the Vaccine Safety Datalink*, 29 VACCINE 8279 (2011).

[77] Lisen Arnheim-Dahlström, *Autoimmune, Neurological, and Venous Thromboembolic Adverse Events After Immunisation of Adolescent Girls with Quadrivalent Human Papillomavirus Vaccine in Denmark and Sweden: Cohort Study*, 347 BMJ 1 (2013).

[78] Allison L. Naleway et al., *Absence of Venous Thromboembolism Risk Following Quadrivalent Human Papillomavirus Vaccination, Vaccine Safety Datalink*, 34 VACCINE 167 (2016).

[79] C. Chao et al., *Surveillance of Autoimmune Conditions Following Routine use of Quadrivalent Human Papillomavirus Vaccine*, 271 J. INTERNAL MED. 193 (2011).

[80] Nikolai Madrid Scheller et al., *Quadrivalent HPV Vaccination and Risk of Multiple Sclerosis and Other Demyelinating Diseases of the Central Nervous System*, 313 JAMA 54 (2015).

[81] C. Genovese et al., *HPV Vaccine and Autoimmune Diseases: Systematic Review and Meta-Analysis of the Literature,* 59 J. PREVENTIVE MED. HYGIENE 194 (2018).

[82] Jozica Skufca et al., *The Association of Adverse Events with Bivalent Human Papilloma Virus Vaccination: A Nationwide Register-Based Cohort Study in Finland,* 36 VACCINE 5926 (2018).

[83] Soraya Azzawi et al., *Immune Privilege Collapse and Alopecia Development: Is Stress a Factor*, 4 SKIN APPENDAGE DISORDERS 236 (2018).

ECF No. 60-6;[84] Resp't's Ex. C, Tab 37, ECF No. 60-7).[85] Dr. Senna cited a 2017 World Congress of Hair Research article[86] that tracked "77 [AA] patients seen over a 10-month period [and] found that 63% of patients cited a stressor as an inciting factor for AA disease exacerbation." *Id.* (citing Resp't's Ex. C, Tab 42, ECF No. 61-2). She continued, "[s]tressors included personal illness/infection, death of a loved one, and work or relationship stress. The duration between the timing of the stressor and the onset or exacerbation in AA was [two to three] months." *Id.* In the present case, Dr. Senna identified family circumstances as Petitioner's potential stressor, including "his maternal grandmother['s] bladder cancer [diagnosis] at a time that his mother was teaching and in graduate school." *Id.* at 14. She further stated that "[a]lthough [Petitioner] is clearly a resilient young man, it is not infrequent that stressors like this contribute to hair follicle immune collapse in genetically predisposed patients, even in the most stoic pediatric patients." *Id.*

Finally, Dr. Senna stated that Petitioner's "AA treatment regimen was conservative at best and lacked an evidence-based approach." *Id.* She described how Petitioner sought treatment for his AA with acupuncture, herbal supplements, and a vitamin B12 injection; however, "[n]one of these treatment modalities have been shown to be beneficial to reversing the inflammation in [AA]." *Id.* Dr. Senna argued that the "best therapeutic interventions for this condition were not implemented early enough and likely contributed to the progression of his [AA] from patchy involvement to more severe involvement." *Id.* at 15–16.

4. Respondent's Expert, Dr. Maverakis

Dr. Maverakis agreed with Dr. Senna's assessment that, given the vast number of children that are vaccinated in the United States versus the incidence rate of AA, "by chance alone, we expect there to be thousands of AA cases occurring in children after vaccination, or any other random predetermined event." Resp't's Ex. H at 4, ECF No. 86-1 . He also agreed with Petitioner's diagnosis of severe AA; however, he noted that "[t]here is no strong reason to believe that [Petitioner's] AA developed because he was vaccinated." *Id.* Dr. Maverakis then discussed the Wise et al. article, which he described as "not specific for AA, as all causes of hair loss were included." *Id.* (citing Resp't's Ex. H, Tab 12, ECF No. 86-13). Furthermore, Dr. Maverakis distinguished the argument made in that paper from the current case, noting the transient hair loss discussed, "is unlike the current case." *Id.*

There was also an agreement between Drs. Maverakis and Dr. Senna that "the ability of [the HPV vaccine] to induce autoimmunity has been specifically and extensively studied." *Id.* at 5. Dr. Maverakis opined that "studies have uniformly demonstrated that there is no association between autoimmune diseases and HPV vaccination." *Id.* Of note to Dr. Maverakis, is the Gronlund et al.[87] study wherein "a cohort of patients with one form of autoimmunity were

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[84] F. Rajabi et al., *Alopecia Areata: A Review of Disease Pathogenesis*, 179 BRIT. J. DERMATOLOGY 1033.
[85] R. Paus, *Exploring the "Brain-Skin Connection": Leads and Lessons from the Hair Follicle*, 64 CURRENT RSCH. TRANSLATIONAL MED. 207 (2016).
[86] Ralf Paus et al., *Neuroendocrinology of the Hair Follicle: Principles and Clinical Perspectives*, 20 TRENDS MOLECULAR MED. 559 (2014).
[87] O. Grönlund et al., *Incidence of New-Onset Autoimmune Disease in Girls and Women with Pre-Existing Autoimmune Disease After Quadrivalent Human Papillomavirus Vaccination: A Cohort Study*, 280 J. INTERNAL MED. 618 (2016).

monitored for the development of a second form of autoimmunity after receiving the HPV vaccination." *Id.* (citing Resp't's Ex. H, Tab 19, ECF No. 86-20). According to Dr. Maverakis, the increased susceptibility of autoimmune disease patients to additional autoimmune conditions allowed for the monitoring of such patients, both vaccinated and unvaccinated, to track the "development of a new form of autoimmunity [and] determine if a particular exposure, e.g. HPV vaccination, increases the risk of autoimmunity." *Id.* The Gronlund et al. study, which he discussed at length, "not only failed to identify a positive association between the HPV vaccination and autoimmunity, but it showed that patients who had received the HPV vaccination were less likely to develop additional forms of autoimmunity." *Id.* (citing Resp't's Ex. H, Tab 19). Dr. Maverakis asserted that "we can confidently conclude that HPV vaccination does not predispose individuals with autoimmunity to develop new onset autoimmunity." *Id.* He conceded that the studies he relied on do not specifically mention AA, however, he noted that "several AA-related autoimmune diseases were indeed included." *Id.*

Dr. Maverakis noted that "[t]he pathogenic immune response in AA has been extensively studied[,] and NKG2D+ CD8+ T cells appear to be the main drivers" of the disease. *Id.* at 6. Dr. Maverakis reiterated Dr. Levinson's point that "in the majority of vaccinated individuals, vaccine-specific CD8+ T cell responses are not detectable." *Id.* (citing Resp't's Ex. H, Tab 23, ECF No. 86-24;[88] Resp't's Ex. H, Tab 24, ECF No. 86-25).[89] Specifically, "[w]hen CD8+ T cell responses to the Gardasil vaccine were specifically studied, CD8 responses were below reliable detection." *Id.* (citing Resp't's Ex. H, Tab 24). He concluded that because the HPV vaccine does not activate the relevant immune cells for AA pathology, "there is no reliable immunological link between the pathophysiology of AA and the immune response induced by HPV vaccination." *Id.*

In response to some of the points Dr. Gershwin made, Dr. Maverakis included a Comment/Reply section at the end of his report. The replies were restatements of arguments previously made by Respondent's other experts and opinions Dr. Maverakis expressed at earlier points in his report. He then reiterated that Petitioner's causation theory is inconsistent with AA pathogenesis, and Petitioner's medical history, including atopy, is evidence of his susceptibility to AA without relation to his HPV vaccination. *See id.* 6–9.

IV. Applicable Legal Standards

I am resolving Petitioner's claim on the filed record. The Vaccine Act and Rules not only contemplate but encourage special masters to decide petitions on the papers where, in the exercise of their discretion, they conclude that doing so will properly and fairly resolve the case. *See* 42 U.S.C. § 12(d)(2)(D); Vaccine Rule 8(d). The decision to rule on the record in lieu of a hearing has been affirmed on appeal. *Kreizenbeck v. Sec'y of Health & Hum. Servs.*, 945 F.3d 1362, 1366 (Fed. Cir. 2020); *see also Hooker v. Sec'y of Health & Hum. Servs.*, No. 02-472V, 2016 WL 3456435, at *21 n.19 (Fed. Cl. Spec. Mstr. May 19, 2016) (citing numerous cases where special masters decided cases on the papers in lieu of hearing and those decisions were upheld). I am simply not required to hold a hearing in every matter, no matter the preferences of the parties. *Hovey v. Sec'y of Health & Hum. Servs.*, 38 Fed. Cl. 397, 402–03 (1997) (determining that the special master

[88] Gounwa Awad et al., *Robust Hepatitis B Vaccine-Reactive T Cell Responses in Failed Humoral Immunity*, 21 MOLECULAR THERAPY: METHODS CLINICAL DEV. 288 (2021).

[89] Douglas M. Herrin et al., *Comparison of Adaptive and Innate Immune Responses Induced by Licensed Vaccines for Human Papillomavirus*, 10 HUM. VACCINES & IMMUNOTHERAPEUTICS 3446 (2014).

acted within his discretion in denying an evidentiary hearing); *Burns v. Sec'y of Health & Hum. Servs.*, 3 F.3d 415, 417 (Fed. Cir. 1993); *Murphy v. Sec'y of Health & Hum. Servs.*, No. 90-882V, 1991 WL 71500, at *2 (Fed. Cl. Spec. Mstr. Apr. 19, 1991).

To receive compensation under the Vaccine Act, a petitioner must demonstrate either that: (1) the petitioner suffered a "Table injury" by receiving a covered vaccine and subsequently developing a listed injury within the time frame prescribed by the Vaccine Injury Table set forth at 42 U.S.C. § 300aa-14, as modified by 42 C.F.R. § 100.3; or (2) that the petitioner suffered an "off-Table injury," one not listed on the Table, as a result of receiving a covered vaccine. *See* 42 U.S.C. §§ 300aa-11(c)(1)(C); *Moberly v. Sec'y of Health & Hum. Servs.*, 592 F.3d 1315, 1321 (Fed. Cir. 2010); *Capizzano v. Sec'y of Health & Hum. Servs.*, 440 F.3d 1317, 1319–20 (Fed. Cir. 2006). Petitioner does not allege a Table injury in this case; thus, he must prove that his injury was caused-in-fact by a Table vaccine.

To establish causation-in-fact, a petitioner must demonstrate by a preponderance of the evidence that the vaccine was the cause of the injury. 42 U.S.C. § 300aa-13(a)(1)(A). A petitioner is required to prove that the vaccine was "not only a but-for cause of the injury but also a substantial factor in bringing about the injury." *Moberly*, 592 F.3d at 1321–22 (quoting *Shyface v. Sec'y of Health & Hum. Servs.*, 165 F.3d 1344, 1352–53 (Fed. Cir. 1999)).

In the seminal case of *Althen v. Sec'y of Health & Hum. Servs.*, the Federal Circuit set forth a three-pronged test used to determine whether a petitioner has established a causal link between a vaccine and the claimed injury. *See* 418 F.3d 1274, 1278–79 (Fed. Cir. 2005). The *Althen* test requires petitioners to set forth: "(1) a medical theory causally connecting the vaccination and the injury; (2) a logical sequence of cause and effect showing that the vaccination was the reason for the injury; and (3) a showing of a proximate temporal relationship between vaccination and injury." *Id.* at 1278. To establish entitlement to compensation under the Program, a petitioner is required to establish each of the three prongs of *Althen* by a preponderance of the evidence. *Id.* "[C]lose calls regarding causation are resolved in favor of injured claimants." *Id.* at 1280. Further, evidence used to satisfy one prong of the test may overlap to satisfy another prong. *Capizzano,* 440 F.3d at 1326.

A petitioner who satisfies all three prongs of the *Althen* test has established a prima facie showing of causation. *Hammitt v. Sec'y of Health & Hum. Servs*., 98 Fed. Cl. 719, 726 (2011). A petitioner who demonstrates by a preponderance of the evidence that he suffered an injury caused by vaccination is entitled to compensation unless the respondent can demonstrate by a preponderance of the evidence that the injury was caused by factors unrelated to the vaccination. *See Althen*, 418 F.3d at 1278; *Knudsen v. Sec'y of Health & Hum. Servs.*, 35 F.3d 543, 547 (Fed. Cir. 1994). In such a case, the government must not merely prove the existence of an alternative cause, but that such an alternative actually caused the injury. *Knudsen,* 35 F.3d at 549. Consequently, when and if the petitioner establishes a prima facie case, the burden then shifts to the government to prove that an alternative cause, unrelated to the administration of the vaccine, was the "sole substantial factor" in causing the alleged injury. *See de Bazan v. Sec'y of Health & Hum. Servs.*, 539 F.3d 1347, 1354 (Fed. Cir. 2008); *see also Hammitt*, 98 Fed. Cl. at 726 (explaining that the respondent's burden is to show that the "factor unrelated" was the "sole substantial factor" in causing the injury). Additionally, a factor unrelated "may not include 'any idiopathic, unexplained, unknown, hypothetical, or undocumentable cause, factor, injury, illness or condition.'" 42 U.S.C. § 300aa-13(a)(2); *see also Doe v. Sec'y of Health & Hum. Servs.*, 601

F.3d 1349 (Fed. Cir. 2010) (stating that an idiopathic diagnosis cannot be a "factor unrelated," as it is idiopathic).

V. Discussion

A. *Althen* Prong One

Under the first prong of *Althen*, a petitioner must offer a scientific or medical theory that answers in the affirmative the question: "can the vaccine[] at issue cause the type of injury alleged?" *See Pafford v. Sec'y of Health & Hum. Servs.*, No. 01-0165V, 2004 WL 1717359, at *4 (Fed. Cl. Spec. Mstr. July 16, 2004), *mot. for rev. denied*, 64 Fed. Cl. 19 (2005), *aff'd*, 451 F.3d 1352 (Fed. Cir. 2006). A petitioner's theory must be based on a "sound and reliable medical or scientific explanation." *Knudsen*, 35 F.3d at 548. Such a theory must only be "legally probable, not medically or scientifically certain." *Id.* at 548–49. Petitioners are not required to identify "specific biological mechanisms" to establish causation, nor are they required to present "epidemiologic studies, rechallenge[] the presence of pathological markers or genetic disposition, or general acceptance in the scientific or medical communities." *Capizzano,* 440 F.3d at 1325 (quoting *Althen*, 418 F.3d at 1280). Scientific and "objective confirmation" of the medical theory with additional medical documentation is unnecessary. *Althen*, 418 F.3d at 1278–81; *see also Moberly*, 592 F.3d at 1322. However, as the Federal Circuit has made clear, "simply identifying a 'plausible' theory of causation is insufficient for a petitioner to meet her burden of proof." *LaLonde v. Sec'y of Health & Hum. Servs.*, 746 F.3d 1334, 1339 (Fed. Cir. 2014) (citing *Moberly*, 592 F.3d at 1322). Indeed, the Federal Circuit has "consistently rejected theories that the vaccine only 'likely caused' the injury and reiterated that a 'plausible' or 'possible' causal theory does not satisfy the standard." *Boatmon v. Sec'y of Health & Hum. Servs.*, 941 F.3d 1351, (Fed. Cir. 2019) (citing *Moberly*, 592 F.3d at 1322 and *LaLonde*, 746 F.3d at 1339). Rather, "[a] petitioner must provide a reputable medical or scientific explanation that pertains specifically to the petitioner's case." *Moberly*, 592 F.3d at 1322. In general, "the statutory standard of preponderance of the evidence requires a petitioner to demonstrate that the vaccine more likely than not caused the condition alleged." *LaLonde,* 746 F.3d at 1339.

Presently, Petitioner does not establish that it is more likely than not that the HPV vaccine can cause AA. His theory that molecular mimicry between viral antigens in Gardasil and self-antigens expressed on the hair follicle led to the development of AA is not supported by sufficient reliable evidence. In his expert report, Dr. Gershwin opined that "[t]he mechanism that leads to alopecia includes [] genetic susceptibility and the generation of cytotoxic CD8 T cells." Pet'r's Ex. 16 at 5. Specifically, he asserted that the vaccine "produced cytotoxic T cells that were directed at [Petitioner's] hair follicles and which cross react with an epitope or region of the Gardasil vaccine." *Id.* This iteration of molecular mimicry begins with the HPV vaccine and ends with AA. For this sequence to compose a successful theory, all these pieces must work together, even if it is not quite understood how. Petitioner did not present reliable evidence of the assumed intermediate step: the HPV vaccine's role in the production of CD8+ T cells. This is where the theory fails.

While Petitioner does not need to rely on medical literature specifically to support his theory, he must still provide some form of preponderant evidence that his proposed mechanism can or does occur. Dr. Gershwin filed four different expert reports with accompanying medical literature but did not identify persuasive support for his contention that the Gardasil vaccine induces the production of cytotoxic T cells.

Dr. Gershwin cited an article from Colmenares et al. to assert that this literature "notes the imbalance of CD8 T cells following Gardasil vaccine." Pet'r's Ex. 99 at 2. The imbalance that the authors observed was a decrease in certain CD8+ lymphocytes, but an increase "in the density (MFI) of [the ILT2] receptor in these cells." Pet'r's Ex. 105 at 7. The article does not discuss how this imbalance is related to AA pathology or autoimmune disease in general. In fact, the authors viewed their findings as indications "that HPV immunization is associated with significant changes in the expression and function of immune innate and regulatory receptors, phenomena that may contribute to the protective effect of this vaccine." *Id.* The article did not address Dr. Gershwin's contention that HPV vaccination leads to the production of CD8+ cytotoxic cells.

Dr. Gershwin acknowledged the de minimis effect that the addition of an aluminum adjuvant has on cellular mediated immunity. However, he asserted that the addition of polysorbate 80 to Gardasil enables the vaccine to elicit not only stronger cellular immunity but also the production of cytotoxic CD8+ T cells. *See* Pet'r's Ex. 106 at 1–2. Dr. Levinson disputed Dr. Gershwin's opinion and asserted that "the principle immune response elicited by an aluminum compound-[adjuvanted] vaccine is essentially an antibody response." Resp't's Ex. E at 3. Dr. Levinson further stated that "vaccines [adjuvanted] with aluminum salts, are very effective generating potent antibody responses but are ineffective generating protective cell-mediated immune responses, especially cytotoxic T cell responses." *Id.* Dr. Maverakis similarly noted that "[t]raditional vaccines predominantly activate CD4 T cells and B cells. CD8 T cells are not strongly activated by traditional vaccines." Resp't's Ex. H at 7. Respondent's argument is more persuasive. Dr. Gershwin's response that Dr. Levinson's argument is not supported by "a single paper that states that CD8 responses do not occur or are not possible," highlights the same problem for his argument. Pet'r's Ex. 116 at 1. There is no medical literature that provides persuasive support for Dr. Gershwin's contention that the Gardasil vaccine results in the production of cytotoxic T cells. Respondent does not have the burden of proof unless and until Petitioner presents a prima facie case. In this case, Petitioner did not meet this burden.

Dr. Gershwin further pointed to the Macleod et al. publication and asserted that there exists an immunomodulatory molecule in Gardasil that enables this vaccine with the capacity to induce functionally active CD8+ T cells in an immunized host. *See* Pet'r's Ex. 106 at 1 (citing Pet'r's Ex. 108). However, this publication investigated the immunomodulatory agent, MPLA, and did not connect immunomodulatory agents within the Gardasil vaccine to the development of cytotoxic T cells. The article specifically discussed influenza vaccinations, and the authors stated, "effective cytotoxic T-cell differentiation occurred only in the presence of [aluminum and] an additional adjuvant, [MPLA]." Pet'r's Ex. 108 at 1. Dr. Gershwin referred to this article to show that certain agents, when added to a vaccine, can promote cytotoxic T cells. He further stated that "[t]he use of other materials in vaccines, when alum is included, to facilitate cell-mediated immunity has been known for decades," but ultimately failed to point to MPLA or another immunomodulatory agent within the Gardasil vaccine that produces cytotoxic T cells. Pet'r's Ex. 106 at 2 (citing Pet'r's Ex. 112).

In one of his supplemental reports, Dr. Gershwin clarified his opinion that Petitioner "would not have developed alopecia if he did not uniquely have an antigen in his hair follicles that was cross-reactive with a component of the HPV vaccine." *Id.* Thus, he claimed, this self-antigen that was always present was the basis for the "continued production of cytotoxic CD8 T cells and subsequent alopecia." *Id.* Dr. Gershwin asserted that this self antigen is a necessary condition for the development of AA following HPV vaccination; however, he does not provide preponderant

evidence of the presence of a unique antigen that is distinct from the generally accepted genetic predisposition that some individuals have to autoimmune disease. Furthermore, the studies that Dr. Gershwin cited did not mention AA at all. In fact, there was no connection made between exposure to any vaccination and the development of an autoimmune disease due to the production of CD8+ T cells.

In *Farag* and *Cordova*, Dr. Gershwin was also the petitioners' expert asserting a causal relationship between the HPV vaccine and AA. *Farag v. Sec'y of Health & Hum. Servs.*, No. 17-714V, 2023 WL 7203034 (Fed. Cl. Spec. Mstr. Sept. 29, 2023); *Cordova v. Sec'y of Health & Hum. Servs*., No. 17-1282V, 2021 WL 3285367 (Fed. Cl. Spec. Mstr. June 21, 2021). Similar to this case, in *Farag,* the petitioner did not file "a single article indicating that the HPV vaccine can or does produce cytotoxic T cells." 2023 WL 7203034, at *20. I concluded that the petitioner failed to present preponderant evidence that the HPV vaccine could cause AA via the upregulation of cytotoxic T cells. *Id.* In *Cordova*, the petitioner presented "a few small-scale studies aimed at evaluating the immunogenicity of the HPV vaccine, noting that they suggest some increased T cell upregulation after receipt of the vaccine." 2021 WL 3285367, at *17. Nonetheless, the Chief Special Master ultimately found that the articles did not show an increase in T cells that was "(a) []likely causal of AA, or (b) even involve[] the specific kind of cytotoxic T cells that drive AA." *Id.* Petitioner was unsuccessful in this case as well. Although special masters are not bound by self-authored prior decisions or the decisions of other special masters, these cases can be helpful in the evaluation of evidence that has been previously introduced and considered in the Program.

Dr. Levinson asserted that "there is no evidence in the extant scientific literature that supports the contention that any type of molecular mimicry accounts for the autoimmune destruction of hair cells in AA." Resp't's Ex. E at 1. In her expert report, Dr. Senna explained that there was no evidence that the HPV vaccine caused AA or autoimmune disease. *See* Resp't's. Ex. C at 11. Her report relied on data analysis, where "[a] large meta-analysis on six studies on bivalent and quadrivalent HPV vaccines showed that in 243,289 patients who received the HPV vaccines there was no correlation between autoimmune disorders and HPV vaccination." *Id.*; *see* Resp't's Exhibit C, Tab 50. Further, she cited VAERS to state that no reports of AA or other forms of hair loss were reported in females and males who received HPV vaccination between 2009 through 2015. *Id.* (citing Resp't's Ex. C, Tab 52, ECF No. 62-2).[90] In Dr. Maverakis' report, he noted that "[t]here is no evidence that HPV vaccination causes, or significantly aggravates, preexisting autoimmunity. In fact, when at risk individuals were studied, HPV vaccination was associated with a reduced risk of autoimmunity, i.e., HPV vaccination was protective for autoimmunity." Resp't's Ex. H at 9. He then pointed to the Gronlund et al. study, which "not only failed to identify a positive association between HPV vaccination and autoimmunity, but it showed that patients who had received the HPV vaccination were less likely to develop additional forms of autoimmunity." *Id.* at 5 (citing Resp't's Ex. H, Tab, 19). Due to the rare occurrence rate of many of the injuries that are examined in the Vaccine Program, it is often difficult, if not impossible, to find relevant studies to prove or disprove vaccine causation. Consequently, this type of evidence is not required to bring a successful claim. However, to the extent that studies do exist, they can be used to support or rebut a theory that a petitioner presents. In the present case, the vaccination is widespread, and the disease incident rate is over one percent of the population. Respondent's experts have persuasively

[90] Jorge E. Arana et al., *Post-Licensure Safety Monitoring of Quadrivalent Human Papillomavirus Vaccine in the Vaccine Adverse Event Reporting System (VAERS), 2009-2015*, 36 VACCINE 1781 (2018).

used studies that fail to show a correlation as supporting evidence that the proposed mechanism is faulty. Dr. Gershwin is not required to identify large scale studies or conclusive literature. However, he failed to persuasively rebut the arguments and support provided by Respondent's experts. Petitioner must do more than demonstrate a 'plausible' or 'possible' causal link between the vaccination and the injury. *W.C. v. Sec'y of Health & Hum. Servs.,* 704 F.3d at 1356 (Fed. Cir. 2013).

Dr. Gershwin presented the Tu et al. article to show "increased incidence of [AA] after HPV infection." Pet'r's Ex. 116 at 1 (citing Pet'r's Ex. 118). His reliance on this article does not provide preponderant support for his proposition because the article "reviewed patient medical records spanning a period of approximately twelve years, and they did not indicate when the patients in the HPV group developed AA relative to the onset of their HPV symptoms or the date they were infected with HPV." *Farag*, 2023 WL 7203034 at *22. Further, the results are limited in application because the underlying data is derived from a mono-country evaluation, which may not be applicable to non-Asian ethnic groups, and the authors did not incorporate data regarding HPV vaccination.

Lastly, even if the HPV vaccine could induce a pathologic cytotoxic T cell response, Petitioner has failed to demonstrate by preponderant evidence the second component of his theory, that such a response could destroy the immune privilege of the hair follicle to cause AA. Although Program petitioners often present homologies between vaccine components and human tissues, such is not required to present preponderant support for a theory of molecular mimicry. However, petitioners must present some form of preponderant evidence that a cross reaction between the vaccine and the body part at issue can occur. Indeed, "[p]etitioners cannot simply invoke the concept of molecular mimicry and call it a day. . . . Rather, they need to offer *reliable* and persuasive medical or scientific evidence of some kind . . . that suggests the vaccine components could interact with self structures as maintained." *Johnson v. Sec'y of Health & Hum. Servs.*, No. 14-254V, 2018 WL 2051760, at *26 (Fed. Cl. Spec. Mstr. Mar. 23, 2018). Petitioner has not provided preponderant evidence that the HPV vaccine can cause AA, pursuant to *Althen* prong one.

B. Althen Prong Two

Under the second prong of *Althen*, a petitioner must prove that the vaccine actually did cause the alleged injury in a particular case. *See Pafford*, 2004 WL 1717359, at *4; *Althen*, 418 F.3d at 1279. The second *Althen* prong requires proof of a logical sequence of cause and effect, usually supported by facts derived from a petitioner's medical records. *Althen*, 418 F.3d at 1278; *Capizzano*, 440 F.3d at 1326; *Grant v. Sec'y of Health & Hum. Servs.*, 956 F.2d 1144, 1148 (Fed. Cir. 1992). A petitioner does not meet this obligation by showing only a temporal association between the vaccination and the injury; instead, the petitioner "must explain *how* and *why* the injury occurred." *Pafford*, 2004 WL 1717359, at *4 (emphasis in original). The special master in *Pafford* noted petitioners "must prove [] both that her vaccinations were a substantial factor in causing the illness . . . and that the harm would not have occurred in the absence of the vaccination." 2004 WL 1717359, at *4 (citing *Shyface*, 165 F.3d at 1352). A reputable medical or scientific explanation must support this logical sequence of cause and effect. *Hodges v. Sec'y of Health & Hum. Servs.,* 9 F.3d 958, 961 (Fed Cir. 1993) (citation omitted). Nevertheless, "[r]equiring epidemiologic studies . . . or general acceptance in the scientific or medical communities . . . impermissibly raises a claimant's burden under the Vaccine Act and hinders the

system created by Congress . . . .” *Capizzano*, 440 F.3d at 1325–26. “[C]lose calls regarding causation are resolved in favor of injured claimants.” *Althen*, 418 F.3d at 1280.

In Program cases, contemporaneous medical records and the opinions of treating physicians are favored. *Capizzano*, 440 F.3d at 1326 (citing *Althen*, 418 F.3d at 1280). Indeed, when reviewing the record, a special master must consider the opinions of treating physicians. *Capizzano*, 440 F.3d at 1326. This is because “treating physicians are likely to be in the best position to determine whether ‘a logical sequence of cause and effect show[s] that the vaccination was the reason for the injury.’” *Id.* In addition, “[m]edical records, in general, warrant consideration as trustworthy evidence. The records contain information supplied to or by health professionals to facilitate diagnosis and treatment of medical conditions. With proper treatment hanging in the balance, accuracy has an extra premium. These records are also generally contemporaneous to the medical events.” *Cucuras v. Sec’y of Health & Hum. Servs.*, 993 F.2d 1525, 1528 (Fed. Cir. 1993). While a special master must consider these opinions and records, they are not “binding on the special master or court.” 42 U.S.C. § 300aa-13(b)(1). Rather, when “evaluating the weight to be afforded to any such . . . [evidence], the special master . . . shall consider the entire record . . . .” *Id.*

Petitioner has not established a logical sequence of cause and effect between his vaccination and his AA. In his initial expert report, Dr. Gershwin asserted that “the kinetics of appearance of cytotoxic T cells after vaccination can occur within several days.” Pet’r’s Ex. 16 at 5. In Petitioner’s case, Dr. Gershwin opined that “[o]nset within 14 days would certainly be consistent with generation of a CD8 response,” and that there was no other immunological challenge to Petitioner’s system during this period. *Id.* In a subsequent report, Dr. Gershwin argued that Petitioner “would not have developed alopecia if he did not uniquely have an antigen in his hair follicles that was cross-reactive with a component of the HPV vaccine.” Pet’r’s Ex. 106 at 2. A review of Dr. Gershwin’s four expert reports reveals brief discussions of Petitioner’s specific facts and circumstances to establish a logical sequence of cause and effect. While petitioners’ experts usually find support for specific causation within a petitioner’s medical records, there is no requirement that medical records must be used to establish that a specific vaccine caused a specific injury in a specific person. Here, Petitioner has failed to present preponderant evidence that he experienced an autoimmune reaction following any of his HPV vaccinations. While this evidence is not mandatory, evidence of such a reaction could support Petitioner’s contention that his vaccination caused autoimmunity, which then led to his injury. Of note is Dr. Senna’s observation that “[Petitioner] was not reported to experience even the most commonly reported adverse effects after any of his [three] HPV vaccines, including local injection site reactions, syncope, dizziness or other symptoms.” Resp’t’s Ex. C at 12. Petitioner’s remaining argument for causation would be based solely on temporal proximity. It is well established in the Program that temporal proximity between a vaccination and injury is insufficient to support causation. *Moberly*, 592 F.3d at 1323 (quoting *Althen*, 418 F.3d at 1278) (“[N]either a mere showing of a proximate temporal relationship between vaccination and injury, nor a simplistic elimination of other potential causes of the injury suffices, without more, to meet the burden of showing actual causation.”); *Sumner v. Sec’y of Health & Hum. Servs.*, No. 99-946V, 2015 WL 5173644, at *9 (Fed. Cl. Spec. Mstr. Aug. 13, 2015) (“[W]here a petitioner’s expert views the temporal relationship as the ‘key’ indicator of causation, the claim must fail.”).

Ultimately, Dr. Gershwin was unable to identify anything in Petitioner’s medical history besides temporal proximity to connect his vaccination and injury. *See Cordova*, 2021 WL 3285367,

at *18 (finding that the petitioner did not satisfy Prong Two because "only a temporal association links his AA onset to the first HPV dose . . . [and because t]here is no evidence of any immediate reaction or measured autoimmune/inflammatory process that preceded onset."). In fact, he did not identify key symptoms, test results, or other evidence that, if present, would signal an adverse reaction to the HPV vaccine leading to the development of AA. The fact that Petitioner's AA began post vaccination is not itself enough to meet the burden articulated as *Althen* prong two. *See id.*

Dr. Gershwin's opinion also noted that "[t]he only immunological challenge within that window was the HPV vaccination." Pet'r's Ex. 16 at 5. However, Dr. Senna noted that a number of potential triggers and risk factors besides the HPV vaccine may have played a role in this case, including "a history of atopy (allergic rhinitis, seasonal allergies), development at a young age (age 13), and AA affecting the occiput scalp or the ophiasis pattern." Resp't's Ex. C at 12. She further noted that Petitioner "had [three] of the total [eight] known associated risk factors for poor AA prognosis, [which] unequivocally suggests that this is a patient who despite any other exogenous influences is more likely than not to progress to severe [AA]." *Id.* at 13. Dr. Senna identified a specific period in Petitioner's life that may have triggered his AA. Petitioner's "maternal grandmother had been diagnosed with bladder cancer at a time that his mother was teaching and in graduate school." *Id.* at 14. Dr. Senna noted that "[a]lthough [Petitioner] is clearly a resilient young man, it is not infrequent that stressors like this contribute to hair follicle immune collapse in genetically predisposed patients even in the most stoic pediatric patients." *Id.* While petitioners can prevail on Prong Two without necessarily addressing alternative causes, the existence of possible alternative causes underlies why temporal proximity alone is insufficient. *See Winkler v. Sec'y of Health & Human Servs.,* 88 F.4th 958 (Fed. Cir. 2023). Petitioner has not provided preponderant evidence that Petitioner's HPV vaccination caused him to develop AA, pursuant to *Althen* prong two.

C. Althen Prong Three

To satisfy the third *Althen* prong, a petitioner must establish a "proximate temporal relationship" between the vaccination and the alleged injury. *Althen*, 418 F.3d at 1281. This "requires preponderant proof that the onset of symptoms occurred within a timeframe for which, given the medical understanding of the disorder's etiology, it is medically acceptable to infer causation-in-fact." *de Bazan*, 539 F.3d at 1352. Typically, "a petitioner's failure to satisfy the proximate temporal relationship prong is due to the fact that onset was too late after the administration of a vaccine for the vaccine to be the cause." *Id.* However, "cases in which onset is too soon" also fail this prong; "in either case, the temporal relationship is not such that it is medically acceptable to conclude that the vaccination and the injury are causally linked." *Id.*; *see also Locane v. Sec'y of Health & Hum. Servs.*, 685 F.3d 1375, 1381 (Fed. Cir. 2012) ("[If] the illness was present before the vaccine was administered, logically, the vaccine could not have caused the illness.").

Although a temporal association alone is insufficient to establish causation, under the third prong of *Althen*, a petitioner must show that the timing of the injury fits with the causal theory. *See Althen*, 418 F.3d at 1278. The special master cannot infer causation from temporal proximity alone. *See Thibaudeau v. Sec'y of Health & Hum. Servs.*, 24 Cl. Ct. 400, 403–04 (1991); *see also Grant*, 956 F.2d at 1148 ("[T]he inoculation is not the cause of every event that occurs within the ten[-]day period . . . [w]ithout more, this proximate temporal relationship will not support a finding of causation." (quoting *Hasler v. United States*, 718 F.2d 202, 205 (6th Cir. 1983))).

Dr. Gershwin stated that "the kinetics of appearance of cytotoxic T cells after vaccination can occur within several days[.]" Pet'r's Ex. 16 at 5. He then opined that the "onset within l4 days would certainly be consistent with generation of a CD8 response." *Id.* Thus, he concluded that "[b]ased on the literature, the kinetics herein are a medically appropriate time frame from exposure to antigen challenge and the development of autoimmunity based upon on genetic susceptibility and the generation of CD8 cytotoxic cells." *Id.* However, the medical evidence and affidavits in this case are inconsistent with Dr. Gershwin's theory. Petitioner received his third Gardasil vaccination on March 30, 2015, and according to Petitioner's mother, she began to notice an "unusual amount of hair in [Petitioner's] bathroom" around "mid-late April" Pet'r's Ex. 13 at ¶ 7. She then noticed a small ball spot on Petitioner's head on or around May 4, 2015. Petitioner's symptoms were noticed at the earliest, 22 days post vaccination, and at the latest, 32 days post vaccination. While Dr. Gershwin's theory is based on onset within 14 days of vaccination, there is no evidence that Petitioner's AA developed within 14 days. Petitioner has not provided preponderant evidence that Petitioner's onset of AA is consistent with the timeframe articulated by Dr. Gershwin's causation theory, pursuant to *Althen* prong three.

VI. Conclusion

After a careful review of the record, Petitioner has failed to prove by preponderant evidence that his AA was caused-in-fact by his HPV vaccination. Accordingly, I **DENY** Petitioner's claim and **DISMISS** his petition.[91]

IT IS SO ORDERED.,

s/Herbrina D.S. Young
Herbrina D.S. Young
Special Master

[91] Pursuant to Vaccine Rule 11(a), entry of judgment is expedited by the parties' joint filing of a notice renouncing the right to seek review.